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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/672,515	09/25/2003	Peter Adorjan	5013.1022cip	1178

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EXAMINER	
NEGIN, RUSSELL SCOTT	
ART UNIT	PAPER NUMBER
1631	

SHORTENED STATUTORY PERIOD OF RESPONSE	MAIL DATE	DELIVERY MODE
3 MONTHS	03/26/2007	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

If NO period for reply is specified above, the maximum statutory period will apply and will expire 6 MONTHS from the mailing date of this communication.

Office Action Summary	Application No.	Applicant(s)	
	10/672,515	ADORJAN ET AL.	
	Examiner	Art Unit	
	Russell S. Negin	1631	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *12 December 2006*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-52* is/are pending in the application.

4a) Of the above claim(s) *18-24,26-43 and 45-47* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-11,48-50 and 52* is/are rejected.

7)☒ Claim(s) *2,12-17,25,44 and 51* is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Comments

Claims 1-17, 25, 44, and 48-52 are examined on the merits in this Office action.

Claim Objections

The objections to claims 3-6 to because of the informalities are withdrawn due to amendments made by applicants to the set of claims filed on 12 December 2006.

Claim 2 is objected to because of the following informalities:

The status identifier to claim 2 is enclosed in double parentheses.

Appropriate correction is required.

Allowable Subject Matter

Claims 12-17, 25, 44, and 51 are objected to as being dependent upon a rejected base claim, but would be allowable if rewritten in independent form including all of the limitations of the base claim and any intervening claims.

Claim Rejections - 35 USC § 112

The rejections of claims 1-17, 25, 44, and 48-52 under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the

subject matter which applicant regards as the invention, are withdrawn due to arguments made by applicant on page 11 of the Remarks of 25 September 2006.

Claim Rejections - 35 USC § 102

The rejections of claims 1-4, 6-10, and 48 under 35 U.S.C. 102(b) as being anticipated by Janousek et al. [Molecular and General Genetics, vol. 250, pp. 483-490, 1996] are withdrawn due to amendments made by applicant to the specification filed on 12 December 2006.

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

Claims 1-10 and 48 are rejected under 35 U.S.C. 102(b) as being anticipated by Tornaletti et al. [Oncogene, 1995, volume 10, pages 1493-1499].

Claims 1-10 and 48 state:

1. A method for selecting epigenetic features, comprising the steps of:
a) collecting and storing biological samples containing mammalian genomic DNA;
b) collecting and storing available phenotypic information about the biological samples so as to define a phenotypic data set;
c) defining at least one phenotypic parameter of interest;
d) dividing the biological samples into at least two disjunct phenotypic classes of interest using the defined phenotypic parameters of interest;
e) defining an initial set of epigenetic features of interest;
f) analysing the defined epigenetic features of interest of the biological samples so as to generate an epigenetic feature data set;
g) selecting relevant epigenetic features of interest and/or combinations of epigenetic features of interest of the defined epigenetic features of interest, the relevant epigenetic

features of interest and/or combinations of epigenetic features of interest being relevant for epigenetically-based prediction of the at least two phenotypic classes of interest; and h) defining a new set of epigenetic features of interest based on the relevant epigenetic features of interest and/or combinations of epigenetic features of interest generated in step g).

2. The method as recited in claim 1 further comprising repeating steps f) and g) based on the new set of epigenetic features of interest defined in step h).

3. The method as recited in claim 1 wherein the biological samples include at least one of: cells; cellular components which contain DNA; sources of DNA; tissue embedded in paraffin; and histologic object slides.

4. The method as recited in claim 3 wherein the sources of DNA include at least one of: cell lines; biopsies; blood; sputum; stool; urine; and cerebral-spinal fluid.

5. The method as recited in claim 3, wherein the tissue embedded in paraffin includes at least one of tissue from eyes, intestine, kidney, brain, heart, prostate, lung, breast and liver.

6. The method as recited in claim 1 wherein at least one of the phenotypic information and the phenotypic parameter of interest are selected from the group consisting of: kind of tissue; drug resistance; toxicology; organ type; age; life style; disease history; signaling chains; protein synthesis; behavior; drug abuse; patient history; cellular parameters; treatment history; gene expression; and combinations thereof.

7. The method as recited in claim 1 wherein the epigenetic features of interest include cytosine methylation sites in DNA.

8. The method as recited in claim 1 wherein the initial set of epigenetic features of interest is defined using preliminary knowledge data about their correlation with phenotypic parameters.

9. The method as recited in claim 1 wherein the relevant epigenetic feature or a combination of epigenetic features is relevant for epigenetically-based prediction of said phenotypic classes of interest when at least one of an accuracy and a significance of the epigenetically-based prediction of the phenotypic classes of interest is likely to decrease by exclusion of the corresponding epigenetic feature data of the epigenetic feature data set.

10. The method as recited in claim 1 wherein step d) is performed so as to divide the biological samples in two disjunct phenotypic classes of interest.

48. The method as recited in claim 2 wherein the repeating steps f) and g) is performed until a defined number of the epigenetic features of interest and/or combinations of epigenetic features of interest are selected.

The article of Tornaletti et al., entitled, "Complete and tissue-independent methylation of CpG sites in the p53 gene: implications for mutations in human cancers," states in the first sentences of its abstract:

> CpG dinucleotides are the target of about one third of transition mutations found in human genetic diseases and tumors. Methylation at these sites is thought to be the cause of these genetic changes through spontaneous deamination of 5-methylcytosine.

Collection and isolation of human genomic DNA are described in the "Materials and Methods" section on pages 1497-1498 under "DNA and cell culture" and "DNA isolation, base-specific modification and cleavage."

The phenotype selected in this study is the presence or absence of certain types of cancers (two disjunct phenotypic classes of interest). The biological samples are divided into portions specific to each type of cancer phenotype examined. The caption to Figure 1 on Tornaletti et al. states on page 1495:

> Figure 1. Genomic sequencing and methylation analysis of the human p53 gene. The autoradiogram shows the analysis of exon 5, upper strand. Lanes 1-2: C+T- and C-specific Maxam-Gilbert sequencing reactions of unmethylated p53-PCR products; lanes 3-11: C-specific Maxam –Gilbert sequencing reactions of genomic DNA isolated from the following sources: FIB, human skin fibroblasts; KER, normal human epidermal keratinocytes; LUNG, normal human bronchial epithelial cells; MAM, human mammary epithelial cells; COL, normal colonic mucosa cells; BLO, human peripheral blood lymphocytes; HeLa, HeLa S3 cells; CEM, leukemia CEM cells; T-47D, human breast carcinoma cells.

Tornaletti et al. continues in column 2, lines 36-40, by stating:

> Five out of six p53 mutation hotspot codons contain CpG dinucleotides (165, 245, 248, 273, and 282) indicating methylation-driven deamination of 5-mC as a major source of G:C→A:T transition mutations at CpG dinucleotides.

Based on this initial set of methylated CpG dinucleotides, further sets are defined based on iterative autoradiograms of the species of cells mentioned in the caption to Figure 1 of Tornaletti et al. in exons 5-8 of the p53 gene in Figures 1-4, respectively. For example, in exon 1 of Figure 1 of Tornaletti et al., a new set of codons methylated in certain phenotypes- codons 152-153, 156, 158, 170, 175, 181, and 185- are identified. Eliminating a lane from Figure 1 of Tornaletti et al. (i.e. the first or second lanes), makes the process of determining how cytosine methylations of p53 DNA affect the phenotypic outcome of the presence of cancer qualitatively much more difficult. This process described is repeated iteratively for various types of tissue shown in Figure 1 of Tornaletti et al.

Claim Rejections - 35 USC § 103

The rejections of claims 1, 2, 10-16, 25, 44, and 52 under 35 U.S.C. 103(a) as being unpatentable over Janousek et al. [Molecular and General Genetics, vol. 250, pp. 483-490, 1996] in view of Cutis et al. [Annals of Human Genetics, volume 65, pages 95-107, January, 2001] are withdrawn due to amendments made by applicant to the specification filed on 12 December 2006.

The rejections of claims 1, 10, 11, 13, and 17 under 35 U.S.C. 103(a) as being unpatentable over Janousek et al in view of Curtis et al as applied to claims 1, 2, 10-16, 25, 44, and 52 above, and further in view of Spotorno et al [Evolucion Biologica, 1990, volume 4, pages 37-62] are withdrawn due to amendments made by applicant to the specification filed on 12 December 2006.

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

Claims 1, 2, 10-11, 49-50, and 52 are rejected under 35 U.S.C. 103(a) as being unpatentable over Tornaletti et al. in view of Gaasterland et al. [Nature Genetics, March 2000, volume 24, pages 204-206].

Claims 1, 2, 10, 11, 49, 50, and 52 state:

1. A method for selecting epigenetic features, comprising the steps of: a) collecting and storing biological samples containing genomic DNA; b) collecting and storing available phenotypic information about the biological samples so as to define a phenotypic data set; c) defining at least one phenotypic parameter of interest; d) dividing the biological samples into at least two disjunct phenotypic classes of interest using the defined phenotypic parameters of interest; e) defining an initial set of epigenetic features of interest; f) analysing the defined epigenetic features of interest of the biological samples so as to generate an epigenetic feature data set; g) selecting relevant epigenetic features of interest and/or combinations of epigenetic features of interest of the defined epigenetic features of interest, the relevant epigenetic features of interest and/or combinations of epigenetic features of interest being relevant for epigenetically-based

prediction of the at least two phenotypic classes of interest; and h) defining a new set of epigenetic features of interest based on the relevant epigenetic features of interest and/or combinations of epigenetic features of interest generated in step g).

2. The method as recited in claim 1 further comprising repeating steps f) and g) based on the new set of epigenetic features of interest defined in step h).

10. The method as recited in claim 1 wherein step d) is performed so as to divide the biological samples in two disjunct phenotypic classes of interest.

11. The method as recited in claim 10 further comprising performing the epigenetically-based prediction of the at least two phenotypic classes of interest using a machine learning classifier.

49. The method as recited in claim 2, wherein the repeating steps f) and g) is performed until all epigenetic features of interest and/or combinations of epigenetic features of interest of the epigenetic features of interest and/or combinations of epigenetic features of interest with a feature selection criterion score greater than a defined threshold are selected.

50. The method as recited in claim 2, further comprising determining an optimal number of epigenetic features of interest and/or combinations of epigenetic features of interest using crossvalidation of a machine learning classifier on test subsets of epigenetic feature data.

52. The method as recited in claim 1 further comprising training a machine learning classifier using a feature data set corresponding to the defined new set of epigenetic features of interest.

The article of Tornaletti et al., entitled, "Complete and tissue-independent methylation of CpG sites in the p53 gene: implications for mutations in human cancers," states in the first sentences of its abstract:

> CpG dinucleotides are the target of about one third of transition mutations found in human genetic diseases and tumors. Methylation at these sites is thought to be the cause of these genetic changes through spontaneous deamination of 5-methylcytosine.

Collection and isolation of human genomic DNA are described in the "Materials and Methods" section on pages 1497-1498 under "DNA and cell culture" and "DNA isolation, base-specific modification and cleavage."

The phenotype selected in this study is the presence or absence of certain types of cancers (two disjunct phenotypic classes of interest). The biological samples are divided into portions specific to each type of cancer phenotype examined. The caption to Figure 1 on Tornaletti et al. states on page 1495:

> Figure 1. Genomic sequencing and methylation analysis of the human p53 gene. The autoradiogram shows the analysis of exon 5, upper strand. Lanes 1-2: C+T- and C-specific Maxam-Gilbert sequencing reactions of unmethylated p53-PCR products; lanes 3-11: C-specific Maxam –Gilbert sequencing reactions of genomic DNA isolated from the following sources: FIB, human skin fibroblasts; KER, normal human epidermal keratinocytes; LUNG, normal human bronchial epithelial cells; MAM, human mammary epithelial cells; COL, normal colonic mucosa cells; BLO, human peripheral blood lymphocytes; HeLa, HeLa S3 cells; CEM, leukemia CEM cells; T-47D, human breast carcinoma cells.

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Based on this initial set of methylated CpG dinucleotides, further sets are defined based on iterative autoradiograms of the species of cells mentioned in the caption to Figure 1 of Tornaletti et al. in exons 5-8 of the p53 gene in Figures 1-4, respectively. For example, in exon 1 of Figure 1 of Tornaletti et al., a new set of codons methylated in certain phenotypes- codons 152-153, 156, 158, 170, 175, 181, and 185- are identified. Eliminating a lane from Figure 1 of Tornaletti et al. (i.e. the first or second lanes), makes the process of determining how cytosine methylations of p53 DNA affect the phenotypic outcome of the presence of cancer qualitatively much more difficult. This process described is repeated iteratively for various types of tissue shown in Figure 1 of Tornaletti et al.

However, Tornaletti et al. does not use machine learning classifiers to aid in predicting phenotypic information from epigenetic properties.

The study of Gaasterland et al., entitled, "Making the most of microarray data," states in the abstract:

> The impact of microarray technology on biology will depend on computational methods of data analysis. A supervised computer-learning method using support vector machines predicts gene function from expression data—and shows promise.

Gaasterland et al. explain the purpose of using machine learning classifiers in the bottom three columns of page 204:

> Microarray assays can measure the transcriptional effects of changes in gene function under different conditions. They can reveal genes that characterize tissue type, developmental stage, or responses to environmental conditions or genetic modifications. Microarray assays will therefore become a general feature of experimental protocols in genetics and cell physiology. As array data burgeon, new questions arose: if we, as a research community, collect all array hybridization data on a central location, can we assign new genes of unknown function to known functional classes? Can we correlate gene expression with gene function? Can we find new classes of co-regulated genes? Can we extract complete gene regulatory networks from microarray gene expression data?
>
> Computation is our only hope.... Support vector machines (SVMs) a supervised computer-learning method, [is used] to train a 'classification machine' to recognize new genes that are similar in expression pattern to groups of genes that are similar in expression pattern to groups of genes known to be co-regulated.

Figure 1 on page 205 of Gaasterland et al. illustrates the process of machine classification with a threshold. The caption states:

> Fig 1. A support vector machine (SVM) is a computational entity that accepts positive and negative training examples of a topic to be learned. As is 'learns', it draws a hyper-plane [threshold] which maximally separates input data points into two classes, members (green) and non-members (red). Here, input data is shown in three-dimensions...

It would have been obvious at the time of the instant invention for someone of ordinary skill in the art to modify the study of the relation of cytosine methylation to human cancer in view of the machine classification study of Gaasterland et al. because while Tornaletti et al. defined sites on the p53 gene which when methylated result in

human cancer, Gaasterland et al. expands on using machine classifiers to more efficiently and computationally generate accurate predictions of such cancerous phenotypic properties of interest from knowledge of epigenetic features of interest.

Double Patenting

The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. A nonstatutory obviousness-type double patenting rejection is appropriate where the conflicting claims are not identical, but at least one examined application claim is not patentably distinct from the reference claim(s) because the examined application claim is either anticipated by, or would have been obvious over, the reference claim(s). See, e.g., *In re Berg*, 140 F.3d 1428, 46 USPQ2d 1226 (Fed. Cir. 1998); *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) or 1.321(d) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent either is shown to be commonly owned with this application, or claims an invention made as a result of activities undertaken within the scope of a joint research agreement.

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

An obviousness-type double patenting rejection is appropriate where the conflicting claims are not identical, but an examined application claim is not patentably distinct from the reference claim(s) because the examined claim is either anticipated by, or would be obvious over, the reference claim(s). see, e.g., *In re Berg*, 140 F.3d 1428, 46 USPQ2d 1226 (Fed. Cir. 1998); *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985).

Claims 1-2, 6-7, 11-17, 44, and 48-50 are provisionally rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 1-2, 4-5, 9-15, 38, 42-44, respectively, of copending Application No. 10/106,269. Although the conflicting claims are not identical, they are not patentably distinct from each other because the current application limits the classes of phenotypes to disjunct classes while Application No. 10/106,269 does not. However, in the last four lines of page 4 of the specification of copending application 10/106,269, applicants state, "In one particularly preferred embodiment, the phenotypic parameters of interest are used to divide the biological samples in two disjunct phenotypic classes of interest." Thus, disjunct, phenotypic parameters are present in both applications.

In addition, mammalian cells are also present in the example of the reference application. On page 27, under "Experimental protocol," DNA was prepared from 5 human acute myeloid leukemia cell lines. The instant application is generic to the reference in that epigenetic features are not limited to CpG methylations, but the instant application is also specific in that the reference is not limited to mammals or disjunct phenotypic classes of interest. The specification of the reference is consulted as a source to observe if the specific embodiments of the instant application are exemplified by the reference disclosure.

This is a provisional obviousness-type double patenting rejection because the conflicting claims have not in fact been patented.

Response to Arguments

Applicant's arguments filed 12 December 2006 have been fully considered but they are not persuasive.

Applicant's arguments concerning the prior art rejections on pages 12-16 of the Remarks of 25 September 2006 have been considered. In light of the amendments made to the set of claims filed on 25 September 2006, new grounds of rejection have been applied.

Applicant's arguments concerning the double patenting rejection on pages 16-17 of the Remarks of 25 September 2006 have been considered but not found to be persuasive.

Applicant argues that as a result of current amendments, instant claim 1 is not described by copending application 10/106,269. However, although claim 1 of the reference application is generic in the respect of not disclosing mammalian DNA, the reference specification is clear that human cells utilized in the disclosed example.

Conclusion

No claim is allowed.

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Papers related to this application may be submitted to Technical Center 1600 by facsimile transmission. Papers should be faxed to Technical Center 1600 via the central PTO Fax Center. The faxing of such pages must conform with the notices published in the Official Gazette, 1096 OG 30 (November 15, 1988), 1156 OG 61 (November 16, 1993), and 1157 OG 94 (December 28, 1993)(See 37 CFR § 1.6(d)). The Central PTO Fax Center Number is (571) 273-8300.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Russell Negin, Ph.D., whose telephone number is (571) 272-1083. The examiner can normally be reached on Monday-Friday from 7am to 4pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's Supervisor, Irem Yucel, Supervisory Patent Examiner, can be reached at (571) 272-0781.

Information regarding the status of the application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information on the PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

RSN
19 March 2007

19 March 2007

JOHN S. BRUSCA, PH.D
PRIMARY EXAMINER